# A FURTHER CONTRIBUTION TO OUR KNOWLEDGE OF THE HISTOLOGY OF THE GASTRIC MUCOSA IN PATHOLOGICAL CONDITIONS OF THIS ORGAN.*

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IN 1896 I published a paper under a similar title.[1] For the sake of convenience I divided the pieces of gastric mucous membrane that are found in the wash-water or in the chyme according to their microscopic appearance into the following groups:

1. N.—*Normal.* Glands and interglandular tissue exist in normal proportions.
2. C.—*Connective Tissue.* While there is a normal proportion between the glands and interglandular tissue, there is a marked proliferation of connective tissue around the glands.
3. P.—*Proliferation.* There is a marked proliferation of the glands; they are nearer each other, and sometimes have an elongated and curved shape.
4. B.—*Beginning Atrophy.* The glands exist in smaller numbers and are sometimes also smaller in size, the interglandular spaces being quite large and filled partly with small-cell infiltration, partly with connective-tissue formation.
5. A.—*Atrophy.* Complete atrophy; no glands visible; only indications of their previous existence; round-cell infiltration.
6. V.—*Vacuolization.* Within the glands exist vacuoles of different shape, being the result of a mucoid degeneration of some glandular cells.

It is quite evident that this classification does not exhaust all possibilities that may be observed in pathological cases. My idea was only to furnish a foundation which may form a common basis upon which we can build further.

In my former article, to which I will again refer later, I have touched upon the older literature, and will at present cite only the more recent investigations relating to our subject.

Cohnheim,[2] in Boas' clinic, examined a large number of fresh pieces of gastric mucous membrane found in the wash-water, and came to the following conclusions: In cases of hyperacidity parenchymatous changes principally are found, consisting in a disappearance of the principal cells and proliferation of the parietal cells. In cases without hydrochloric acid the principal changes take place in the parenchyma, especially in the vestibular layer. The changes represent a mucoid degeneration of the normal cylindrical cells, and correspond to Hayem's "gastrite parenchymateuse muqueuse"—*i. e.*, the mucous catarrh of authors. In the glandular layer proper we find a disappearance of the parietal cells, with a normal amount of principal cells, as in Case XVIII., in whom the interstitial changes are only slight, and variations from the normal chemistry of the stomach are caused by these. In no case, however, is there a parenchymatous gastritis pure and simple, interstitial changes occurring everywhere to a greater or less degree, beginning with round-cell infiltration and going on to complete atrophy of the glandular layer.

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* Read before the American Gastro-enterological Association, at Washington, D. C., May 1, 1902.

In another paper,[3] dealing more with the diagnosis of cancer of the stomach, the same author arrives at the following conclusions:

1. The only absolutely conclusive sign of cancer that excludes every other doubt is the occurrence of small tumor particles found in washing out either the empty or filled stomach.

2. Nearly as pathognomic—at least according to our present experience—is the development of protozoa on the stomach mucosa.

3. The constant occurrence of pus in fetid or non-fetid stomach contents has up to now been observed only in cancer, although occasionally it may occur when a perigastric abscess breaks into the stomach, but then there is usually no mucus present.

4. All the symptoms may occur before a tumor can be palpated; they are therefore "relatively" early signs.

5. They are principally of importance in the diagnosis of carcinomata of the stomach not situated at the pylorus, since with those situated at the orifice other important signs occur earlier; occasionally, however, they are also observed in these cases.

Strauss and Myer[4] describe a case of hypersecretio continua chronica that came to autopsy. All the glands (both in the fundus and in the pylorus) can be plainly differentiated, but are coiled at their basal extremity. In some places the glands are cystically dilated. Parietal cells are increased. In the connective tissue there is round-cell infiltration, penetrating to the submucosa. In the glandular cells as well as in the gland lumen there are no leucocytes.

Martius and Lubarsch[5] give more exact numbers regarding the distribution of the glands in the stomach. They say in their book:

"I will say right here that in cross-sections of the mucous membrane of the stomach, measured just below its surface, we can see, on an average, twenty to twenty-five glands in one field (Zeiss obj. B.; Oc. 3), the number twenty corresponding about to the pyloric region, where normally the interglandular tissue is somewhat larger than in the other parts of the stomach. Corresponding with these conditions we find that the interglandular connective tissue in its broadest places has a width of five to seven rows of cells at the highest, and these places are found only directly under the surface epithelium. The nearer we approach the gland region proper the less interglandular tissue is found, consisting sometimes only of a few cells, gland lying close to gland. If with the above-mentioned magnification the number of glands is around eighteen, and if there are found ten or more rows of cells in the connective tissue, we can with certainty speak of a proliferation of the connective tissue, even if we are unable to determine whether we have to deal with a piece from the pylorus or the fundus."

Hemmeter[6] has minutely examined the pieces of stomach mucous membrane in a large number of patients. He used my classification given above, and supplemented the same. This author says: "The objection that may be made against this is the fact that Einhorn seemingly had based his classification upon the condition of the glands and the interglandular tissue, regarding the state of the cells themselves only once in his six types. For this reason I will supplement his divisions by adding the appearance and condition of the vestibular cells and the appearance and numerical relations of the principal and parietal cells."

I would like to call attention to the fact that in these examinations we have to deal with small pieces only, where often, especially in cross-sections, it is difficult to say whether only the mouths or the deeper parts of the glands have been cut. For the same reason, numerical data relating to the principal and parietal cells would not be conclusive. Moreover, the significance of the latter has not yet been established.

The result of Hemmeter's investigation was as follows: Ten healthy persons: perfectly normal in eight; proliferation of glands in one; proliferation of connective tissue in one. Hyperacidity in twenty cases: normal in four; atrophy in two; gland proliferation in six; proliferation and hypertrophy of the parietal cells in eight. Anacidity or achylia: normal in two; gland proliferation in one; atrophy in nine.

Leuk[7] gives the following data regarding the relation between the glands and interglandular tissue in the normal human stomach:

In the fundus:

1. The number of gland openings in 1 mm. of mucous membrane (linear measure only) is about sixteen to eighteen.
2. The interfoveal connective tissue lying between two gland openings has a width of about 25 to 30 microns, measured directly under the epithelium.
3. In the middle of the mucous membrane the connective tissue between the glands has a width of 10 to 20 microns.
4. Connective tissue width in the deepest quarter of the mucous membrane, 5 to 10 microns.
5. Width of glands, 30 to 40 microns.

In the pylorus:

1. About nine, distributed regularly.
2. 25 to 30 microns.
3. 25 to 40 microns.

4. In places the same as in the fundus, but between single glands, the connective tissue may be up to 50 microns in width.
5. 45 microns.

Regarding the diagnosis of carcinoma, Lenk expresses himself as follows: "For an absolutely certain diagnosis of carcinoma from small pieces of stomach mucous membrane we must prove an atypical epithelial proliferation from the mucosa into the submucosa. Glands in the submucosa even without mitotic figures must not necessarily be the result of carcinomatous proliferation. Accessory Brunner glands in the pylorus, or simply ends of glands that have been cut off by a branch of the hypertrophic muscularis mucosæ, as I have often seen it in complete sections, might simulate a carcinoma.

"Taking it all in all, I think that our diagnostic knowledge based on the examination of chance pieces of stomach mucous membrane may be enlarged only in especially fortunate cases; in those where we intentionally try to obtain pieces for examination the condition may be somewhat more favorable."

Boekelman[8] examined pieces of stomach mucosa which he was enabled to obtain during gastro-enterostomy. He arrives at the following conclusions:

"All cases of gastric ulcer coincide in the fact that the glands lie closely together, the connective tissue being very narrow. Hyaline bodies are not ordinarily found. Many parietal cells are present, but their number is hard to estimate."

He finds:

1. The openings of the glands of normal length.
2. No goblet cells or ciliary epithelium.
3. Small diameter of the glands.
4. Little or no interstitial tissue.
5. Small round-cell infiltration in the superficial layers.
6. Few or no eosinophile polymorphonuclear leucocytes.
7. Few or no hyaline bodies.
8. Many capillaries and hemorrhages.
9. Many parietal cells, often more than normal.
10. The principal cells usually more affected than the parietal cells.

With carcinoma after ulcer the mucous membrane did not suffer much; in carcinoma without ulcer atrophy was frequently found.

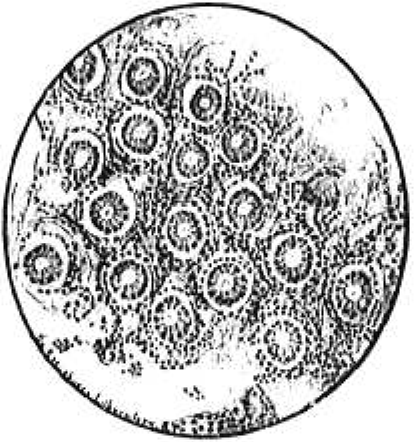
The relation of atrophy to carcinoma ventriculi is described in an older paper by Rosenheim[9] in the following words:

"Atrophic processes in the stomach mucous membrane have a far-reaching influence upon the body economy. They occur more frequently than has been assumed, and not only relatively frequently with carcinoma, but also as a disease of the stomach for itself. They are without doubt important factors in the development of the so-called essential anæmia—more so than they have up to now been credited with." "From our investigations it is evident that atrophy is the result of catarrh and inflammation, and we thus may see the most frequent and benign affection of the stomach converted into a destructive process that threatens life."

Mathieu[10] is also of a similar opinion when he says: "Interstitial gastritis with atrophy of a considerable number of glands is often met with. Not only at the border of the tumor, but also at a distance from it, the glandular elements have most often undergone a retrogressive modification which recalls that seen in gastric polyadenoma." "However that may be, interstitial gastritis and cancer go side by side or follow one another, just as in certain cases of primary cancer of the liver nodular carcinoma and cirrhosis develop simultaneously or successively."

All these authors agree with me in the main that the condition of the stomach mucous membrane in the different secretory states of the stomach is not a constant one.

FIG. 1.



Normal mucous membrane of stomach, showing relation of glands to interglandular substance. Obj. 3; Leitz eye-piece 4.

Having had the opportunity during the last six years, after the publication of my first article, to examine pieces of stomach mucous membrane in a large number of patients, it seemed to me worth while to publish the results of these more recent investigations and to enter more minutely into the details of the histological findings as well as their significance.

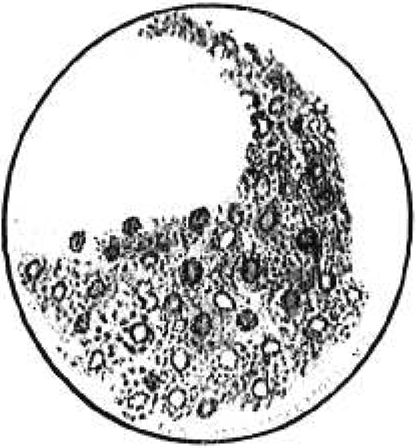
We have tabulated these new cases (thirty-four in all), and designated

## TABLE OF NEW CASES IN WHICH SMALL PIECES OF GASTRIC MUCOSA HAVE BEEN EXAMINED, AND THE RESULT (1896-1902).

| No. | Date. | Name. | Age | Diagnosis. | | Gastric secretory function. | | Anatomical state. | No. glands per sq. millimetre. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Free HCl. | Total acidity. | | | |
| 1 | Feb. 22, 1896 | Mr. H. C. | 45 | Gastritis chronica. | Euchlorhydria. | + | 40 | P. B. | 64 | Atypical glands. |
| 2 | Dec. 30, 1897 | Mr. W. P. | 45 | Dilatatio ventric. ren mobil. dexter. | | + | 44 | P. | 44 | |
| 3 | Oct. 31, 1900 | Mr. C. D. | 55 | Gastritis chronica, hepatitis, icterus. | | + | 46 | P. V. (?) | 80 | Mitoses; piece of submucosa visible. |
| 4 | Nov. 28, 1900 | Mr. W. P. | 41 | Atonia vent., enteroptosis. | | + | 50 | N. V. | 80 | Extravasations of blood; mitoses musc. mucosæ visible. |
| 5 | June 25, 1899 | Mr. W. H. R. | 53 | Hepatitis. | | + | 56 | P. | 68 | Blood extravasations; mitoses; small-cell infiltration; peptic glands. |
| 6 | Dec. 14, 1897 | Mr. Alf. H. | 38 | Ructus, erosions. | | + | 56 | N. | 100 | |
| 7 | April 4, 1898 | Mr. Geo. F. | 42 | Erosions. | Hyperchlorhydria. | + | 64 | P. C. | 72 | Glands small in places; peptic glands. |
| 8 | Oct. 21, 1898 | Mr. E. St. | 27 | Attacks of vomit'g. hyperchlorhydria. | | + | 70 | C. P. | 48 | Large plaques of pavement epithelium. |
| 9 | Feb. 10, 1901 | Mr. B. P. | 36 | Beginning stenosis of pylorus. | | + | 70 | N. P. C. | 88 | Extravasations of red blood cells. |
| 10 | Apr. 29, 1899 | Mr. M. W. | 28 | Dilatatio ventr., ren mob. dext. | | + | 72 | C. P. | 36 | |
| 11 | Apr. 14, 1898 | Miss M. W. | 28 | Gastroptosis, hepatoptosis, ren mob. sin. | | + | 72 | N. C. | 60 | Peptic glands. |
| 12 | Feb. 3, 1896 | Mr. G. A. D. | 32 | Hyperchlorhydria. | | + | 76 | C. P. | 44 | Peptic glands. |
| 13 | ………… | Mr. Z. | 50 | Atonia vent. hyperchlorhydria. | | + | 80 | B. C. | 136 | Glands small (38 microns). |
| 14 | Dec. 21, 1899 | Miss A. E. | 33 | Gastralgia. | | + | 84 | P. C. | | |
| 15 | ………… | Mrs. A. | 44 | Hyperchlorhydria, erosions. | | + | 84 | C. | 76 | Round-cell infiltration in places. |
| 16 | Feb. 20, 1896 | Mr. E. H. C. | 40 | Gastrosuccorrhœa contin. chron. | | + | 92 | P. C. | 128 | |
| 17 | Jan. 30, 1896 | Mr. W. W. S. | 46 | Gastrosuccorrhœa contin. chron. ulcus vent. | | + | 140 | A. | ... | No glands; fine connective tissue, probably from base of ulcer. |
| 18 | Nov. 16, 1901 | Dr. D. | 35 | Enteritis chronica subacidity. | Hypochlorhydria. | + | 26 | P. V. | 160 | Connective tissue scanty; glands atypical. |
| 19 | Nov. 19, 1899 | Dr. O. F. | 42 | Atonia ventric. erosions. | | + | 28 | C. B | 52 | Glands small; connective tissue rich in cells; mitoses; hyaline bodies. |
| 20 | Feb. 23, 1898 | Mrs. F. L. S. | 45 | Dilatatio ventric. | | + | 28 | B. | 60 | Glands small, indistinct; peptic glands. |
| 21 | Feb. 14, 1896 | Mrs. J. S. H. | 35 | Erosions. | | + | 30 | N. B. | 104 | Mitoses (diameter of glands 74 microns.) |
| 22 | Oct. 13, 1900 | Mr. L. C. | 30 | Gastritis chron. erosions. | | + | 30 | C. V. A. | 48 | Cell proliferation; in some cases complete atrophy; numerous capillaries; hemorrhagic infiltrations; hyaline bodies. |
| 23 | Aug. 17, 1901 | Mrs. B. K. | 48 | Dyspepsia nervos. ventriculi. | | ? | ? | N. C. V. | 52 | Some small glands; rich in cellular elements; mitoses; hyaline bodies. |
| 24 | Sept. 14, 1899 | Mr. J. G. D. | 40 | Hyperæsthesia ventric. vomitus, achylia. | Achylia. | 0 | ... | C. | 60 | Extravasations of red blood cells; glands small. |
| 25 | Jan. 14, 1897 | Mrs. J. B. | 60 | Gastritis, enteritis, achylia. | | 0 | ... | P. A. | 40 | Interstitial hemorrhages. |
| 26 | Jan. 27, 1898 | Miss P. F. | 24 | Achylia. | | 0 | ... | B. V. | 120 | Glands small (52 microns). |
| 27 | Jan. 10, 1896 | Mr. D. S. | 48 | Achylia. | | 0 | ... | A. | ... | Some remains of glands. |
| 28 | Nov. 12, 1898 | Mr. D. K. | 34 | Gastritis chron., splenic tumor, carc. ventr. (?) | Carcinoma ventriculi. | 0<br>Rennet + | 12 | B. | 40 | Small round-cell carcinoma? |
| 29 | Jan. 18, 1896 | Mr. M. W. | 42 | Carcinoma vent., ischochymia. | | 0 | 60<br>lactic + | C. | 100 | |
| 30 | Mar. 21, 1898 | Mr. J. D. N. | 65 | Carcinoma vent. et hepatis. | | + | 56 | C. | 44 | Large epithelial cells. |
| 31 | Dec. 4, 1899 | Dr. W. H. | 68 | Carcinoma vent. | | 0 | ... | C. | ... | Large spindle-shaped cells. |
| 32 | Nov. 14, 1899 | Mr. M. K. | 48 | Carcinoma vent. | | 0 | ... | C. | 20 | Glands much enlarged (160 microns), filled with small round cells; epithelium of glands small; mitoses; interglandular hemorrhages. |
| 33 | Jan. 9, 1896 | Mr R. H. A. | 62 | Carcinoma vent., achylia. | | 0 | ... | A. | ... | No glands visible, only traces of destroyed tubules can in some places be discovered. |
| 34 | Sept. 3, 1901 | Mrs. P. | 59 | Carcinoma vent. | | 0 | ... | B. | 32 | Round-cell infiltration; in places more connective tissue; half-destroyed glands; hyaline bodies. |

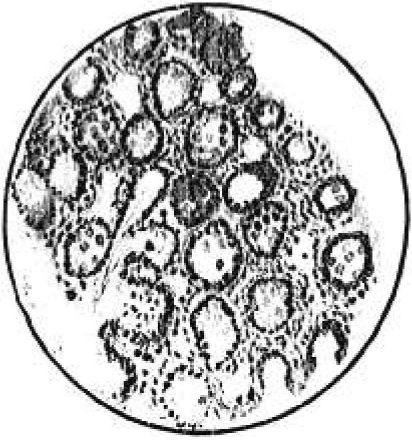
their microscopic appearance by the letters given to the various groups as described above. Before discussing the table we will first

FIG. 2.



* Small glands. (Mr. Z.) Obj. 3; Leitz eye-piece 4.

FIG. 3.

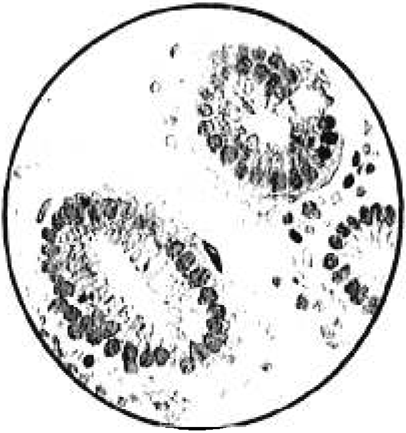


Large dilated glands. (Mr. K.) Obj. 3; Leitz eye-piece 4.

consider some peculiarities of the pathological histology that we have occasionally met with, and illustrate them by means of drawings.

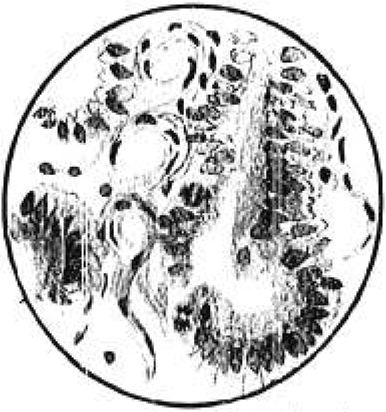
In order to emphasize the presence of abnormal conditions we will first illustrate the appearance of a piece of normal mucous membrane

FIG. 4.



Mitoses, one atypical. (Mrs. J. S. H.) Obj. $^1/_{12}$ oil; Leitz eye-piece 4.

FIG. 5.

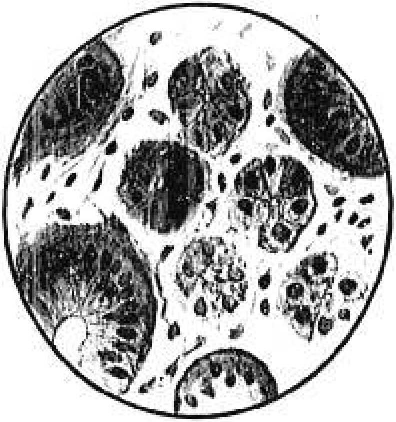


Mitoses, one atypical. (Mr. R.) Obj. $^1/_{12}$ oil; Leitz eye-piece 4.

(Fig. 1), the glands being cut transversely. This specimen was taken from the stomach of a murderer immediately after his execution, and was placed at my disposal through the kindness of Dr. R. Huebner.

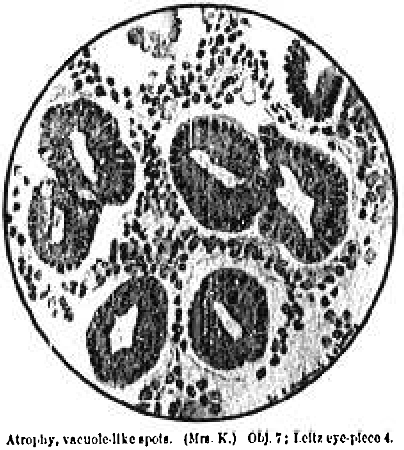
If we take the diameter of a cross-section of a normal stomach gland

FIG. 6.



Parietal cells. (Mr. R.) Obj. $^1/_{12}$ oil; Leitz eye-piece 4.

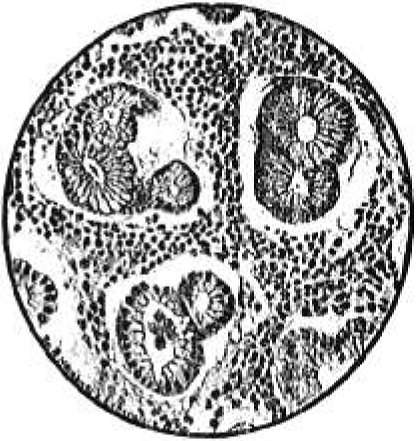
FIG. 7.



Atrophy, vacuole-like spots. (Mrs. K.) Obj. 7; Leitz eye-piece 4.

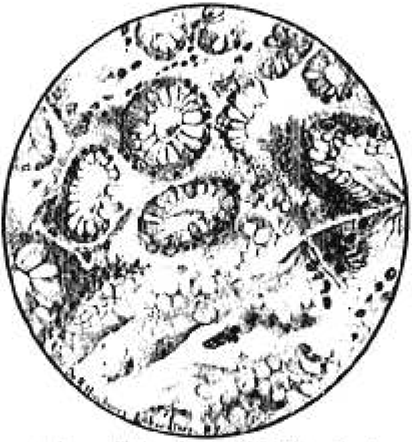
to be about 74 microns, we will find variations from this standard both above and below. Occasionally we observe exceedingly small glands, 38 microns and smaller, occurring sometimes with glands of normal size. They may be found present throughout the whole specimen. (Fig. 2.)

FIG. 8.



Fusion of glands. (Mrs. K.) Obj. 7; Leitz eye-piece 4.

FIG. 9.



Atrophy, vacuolization. (Dr. D.) Obj. 7; Leitz eye-piece 4.

On the other hand, we sometimes find especially large glands (160 microns and larger). The glandular epithelium is usually not enlarged, but there is a marked dilatation of the lumen; we would probably

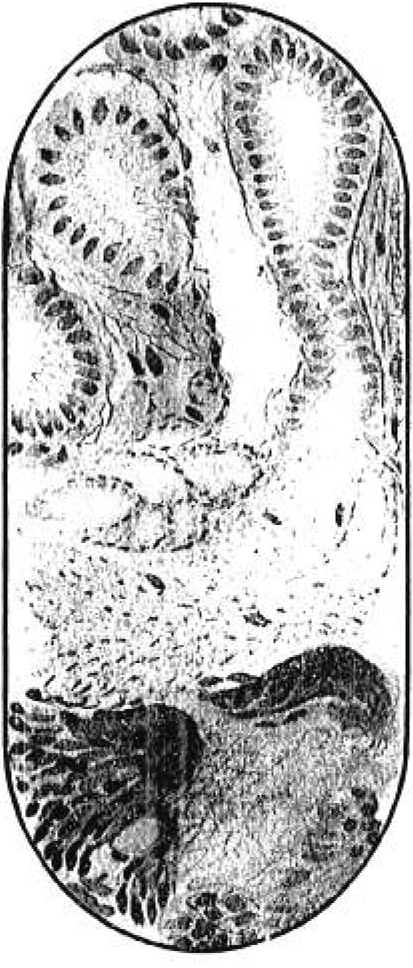
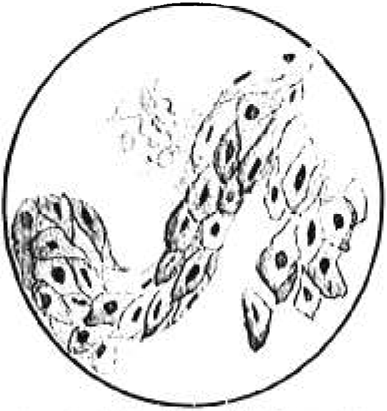


FIG. 10.

A piece of gastric mucosa, showing also submucosa and a portion of the muscular layer of the stomach. (Mr. Wm. P.) Obj. $^1/_{12}$ oil; Leitz eye-piece 4.

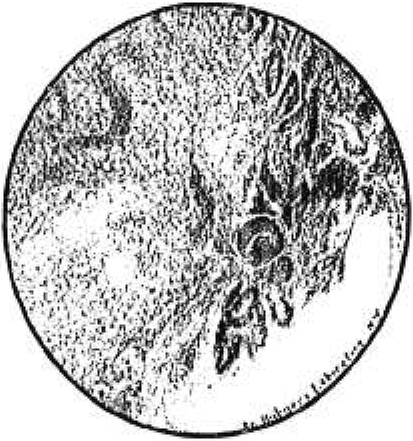
better call them dilated glands. We have met with marked cases of this kind twice. (Fig. 3.) In most places the lumen of the gland is

FIG. 11.



Pavement epithelium. (Mr. R.) Obj. 7; Leitz eye-piece 4.

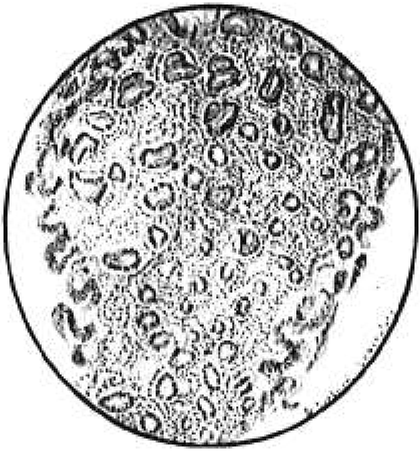
FIG. 12.



Interglandular tissue replaced by epithelial cells, which also invade the glands. (Mr. J. D. N.) Obj. 3; Leitz eye-piece 4.

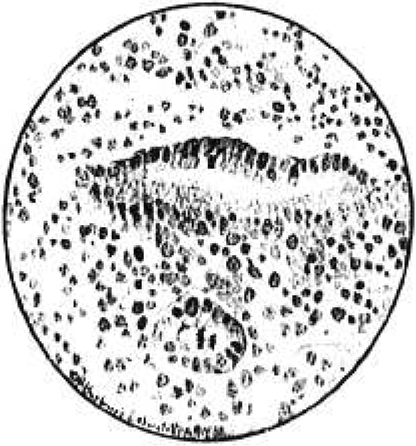
filled with cells, only occasionally the lumen being free, which may be explained by assuming that the contents have dropped out.

FIG. 13.



Atrophy; mitoses; interglandular tissue rich in cells. (Mrs. P.) Obj. 3; Leitz eye-piece 4.

FIG. 14.



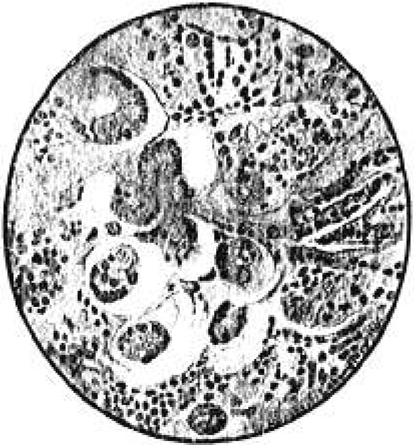
Mitoses. (Mrs. P.) Obj. 7; Leitz eye-piece 4.

An infiltration of leucocytes is often observed in the interglandular structure, more rarely in the glands themselves; the latter condition may be noted in Fig. 4.

Mitoses have been observed in several of the cases; they are by no means typical of neoplasms. Figs. 4 and 5 show karyokineses in typical and atypical form, although in neither case was a neoplasm present.

An increase in the parietal cells of the glands has been observed only a few times. Fig. 6 may serve as an illustration.

FIG. 15.



Atypical glands. (Mrs. P.) Obj. 7; Leitz eye-piece 4.

In some specimens showing evidences of atrophy (increase of interglandular connective tissue, small glands, etc.) we can often observe a kind of blending of the glands, as if they were destroying one another partially. Thus we find two, three, or more glands joined to one another, parts of each gland having disappeared.

In patient K. these conditions are pronounced, and are well shown in Figs. 7 and 8. There are also present in this case vacuole-like formations in the glandular epithelium, stained light blue with hæmatoxylin and showing a fine granulation.

Atrophic conditions may be associated with real vacuolization of the glands. Both conditions are especially marked in the case of Dr. D. (Fig. 9.)

Atrophic processes may also be combined with a tortuous course of the glands and apparent increase of the glands. We find this in the case of Mrs. John B.

The pieces of gastric mucosa that are occasionally found while washing the stomach, and which form the object of our investigations, represent usually only a part of the glandular surface of the stomach. Sometimes, however, the whole mucous membrane, and in rare cases the muscularis mucosæ, or even the submucosa and pieces of the muscular layer attached, are encountered. In the case of W. P., we can plainly observe this rare condition. (Fig. 10.)

Detached shreds of pavement epithelium may be observed off and on in cases that have nothing in common with cancer. Fig. 11 represents such a condition from the specimen of patient R., with slight hyperchlorhydria.

In cases of gastric cancer we may sometimes find signs of a neoplasm in the piece of mucosa, even if the latter does not directly originate from the involved area. In the case of N., with carcinoma ventriculi, there is an extensive epithelial proliferation between the glands. Besides we may be able to demonstrate the invasion of the glands by epithelial (cancer) cells in certain parts destroying the glands. (Fig. 12.)

Patient P., also suffering from cancer of the stomach, shows the signs of atrophy, fusion of the glands (Fig. 13), numerous atypical mitoses and collections of epithelial cells between the glands (Fig. 14); also the encroachment of epithelial cells upon the glands, and, finally, atypical shape of the glands. (Fig. 15.)

(For the beautiful execution of these drawings I am indebted to Dr. R. Huebner.)

Hyaline bodies, or so-called Russell's bodies, have been found in four specimens (one case of carcinoma and three of non-malignant disease). Several of the specimens had been cut too thick, and are unsuitable for the examination of hyaline bodies, otherwise we might have found these elements more frequently. They do not seem to have any pathological significance, although they point to degenerative processes.

If we now return to a consideration of the table we find that for the various secretory states (and also organic diseases) of the stomach there is no constant condition of the mucous membrane—a fact to which we have called attention in our first article.

Even in the group of euchlorhydria (six cases) the structure of the mucosa is not always the same. A proliferation of the glands was found in three, proliferation with beginning atrophy in one, normal in one, and vacuolization in one.

In the eleven cases with hyperchlorhydria we encounter normal conditions of the gland twice; proliferation seven times, either alone or in conjunction with increase of the connective tissue; once beginning atrophy and once absolute glandular atrophy.

Five cases of hypochlorhydria without organic disease showed in one normal conditions, with beginning atrophy; proliferation, with vacuolization, in another; beginning atrophy in two, and increase of connective tissue, with vacuolization, in one.

Four cases of achylia gastrica simplex (without organic lesion) show connective-tissue increase in one; proliferation, with atrophy, in another; beginning atrophy and vacuolization in a third, and complete atrophy in one.

Cases of carcinoma of the stomach: In one case (David K.) the diagnosis of cancer was not absolutely certain, but was very probable; in all the other six the diagnosis of carcinoma at the time of the examination was positive.

In four connective-tissue proliferation was found, in two beginning atrophy, and total atrophy in one. A growth of epithelial cells into the gland lumina was found twice.

From the varying conditions of the mucous membrane of the stomach in the same secretory anomalies we may conclude that the pathologic-anatomical state of this organ is probably not the cause of the affection. In the article above cited I have tried to apply Edinger's" theory for nervous diseases to affections of the stomach. I called attention to the fact that increased functional activity of an organ leads to an increased development of the apparatus kept in activity. Corresponding changes will therefore be produced in the various organs by an increase or decrease of the functional work if continued for any length of time. In the stomach with an increased secretion (hyperchlorhydria, hypersecretion) a glandular proliferation would be expected; whereas, on the other hand, in cases of diminished gastric secretion or total absence of gastric juice (hypochlorhydria, achylia, etc.) a disappearance of the glandular element would be observed.

As a matter of fact, we find this principle indicated on looking at the table. With hyperchlorhydria frequently proliferation is observed, and with hypochlorhydria or achylia frequently beginning or complete atrophy.

The reason why these changes do not always occur I have tried to explain in my former paper by the supposition that the increased or decreased functional activity had perhaps not lasted long enough to produce these changes throughout the entire organ. In addition I would like to remark that, as long as with an increase of functional activity the nutrition of the organ is good, it will hypertrophy; but if the blood supply is inadequate the organ, in spite of increased work, will show the signs of atrophy or degeneration, an exhaustion of some parts occurring, which later perish. In some parts of the stomach similar conditions may at times be present, and thus atrophic states in hyperchlorhydria may be explained.

Why do we find glands in achylia, even if the condition has been present for a long time? Each organ has a certain inherent tendency to retain as much as possible its normal condition, even if not called upon to do much work. The glands will try to perpetuate themselves, even under the most disadvantageous conditions.

According to this explanation, extensive changes in the mucous membrane of the stomach may take place without representing any primary disease of this organ; they may be the result of functional secretory disturbances of long standing.

It is well understood that the pathological processes need not on that account always be of a slight nature, but may show the signs of grave changes.

Although from a diagnostic standpoint the microscopic examination of pieces of mucous membrane of the stomach is inferior to the examination of the gastric juice, yet it may furnish a few prognostically important points. If, for instance, marked proliferation of the glands is found, an existing hyperchlorhydria may be considered to be of a more severe character. In the same way a marked atrophy of the glands in achylia would also be considered of importance.

Martius goes perhaps a little too far when he says: "I believe that as far as the prognosis of a certain stomach trouble is concerned, the result of a histological examination would be of significance. If in a case of achylia gastrica, in spite of subjective euphoria, we should find the anatomical lesions of gastritis progressiva atrophicans, we must bear in mind that a total atrophy of the mucous membrane of the stomach, with subsequent anæmia and cachexia, will be the sad outcome."

I have seen two patients with achylia gastrica (one of whom is David S., in the above table) in whom the examination of the piece of mucous membrane showed a complete atrophy of the glands, and who, in spite of this, have enjoyed the best of health for years. Microscopic examination shows only how far the changes have taken place in the mucous membrane, but gives no hint as to the general bodily condition (how long the body may still exist, etc.). Fortunately, the human organism possesses numerous mechanisms, by means of which even grave functional disturbances with subsequent tissue changes may be compensated.

In carcinoma, also, the examination of pieces of the mucous membrane of the stomach may occasionally be of value, especially when we can demonstrate a direct invasion of the glandular tissue by epithelial cells, as this apparently seems to speak positively for carcinoma.

Footing upon the above theory that the secretory disturbances of the stomach are not dependent upon a primary disease of the mucosa, but are of a purely functional nature, caused by abnormal nerve influences, the following points would suggest themselves in the therapy of these affections:

1. Our therapeutic measures must not be directed against the stomach alone, but must take into consideration the entire bodily state. Any anomalies in the blood or nervous system must first be corrected. Everything must tend to apportion work and rest for the principal organs in an even manner, and bring them into harmony with each other. In this relation the well-known hygienic principles of a correct mode of life are of great importance.

2. Any anomalies should be corrected as far as possible. If this is not possible, everything should be done to make it easy for the anomaly, so that the organism suffers as little as possible.

As to the first point (the improvement of the general bodily condition), we must attempt to establish a correct mode of living, give tonics (iron, arsenic, strychnine, etc.) if needed, or perhaps sedatives for the nervous system (bromides, zinc valerianate, etc.). Hydrotherapeutic measures (massage, gymnastics, general electricity) and change of climate (mountains or seaside) are also of importance.

Regarding the second point (special measures directed against any anomaly of secretion that may be present), we must pay attention to a suitable diet and to direct treatment of the stomach.

In all secretory anomalies as well as in all chronic ailments the principle of giving sufficient nourishment is the main one. We must, therefore, see that the patient eats enough.

In cases of hyperchlorhydria a diet comparatively rich in proteids and poor in carbohydrates will be chosen. Albuminous substances unite with the free hydrochloric acid, thus using up some of the concentrated juice. The subjective feeling of the patient is better, and the strong acid will not so readily injure the mucous membrane of the stomach.

It has been proposed not to give any meat diet in hyperchlorhydria, as this, according to Pawlow, would tend to increase the secretion. We ought, however, not to lose sight of the fact that Pawlow's experiments, interesting as they may be, relate only to normal conditions, and cannot at once be applied to diseased states. In our cases we have to deal with persons whose stomach (probably through abnormal nerve influences) yields a very concentrated stomach juice. Here the meat diet will probably not cause an increased flow of secretion, but rather make good use of the acid, uniting readily with it on the one hand, and on the other hand being more easily digested by the excess of acid. Practically, a diet rich in proteids has stood the test of usefulness in hyperchlorhydria, and there is no reason why we should give it up.

In cases of hypochlorhydria and achylia the food ought to be rich in carbohydrates, and the amount of albumin should be decreased. A fine subdivision of the foodstuffs also is essential here. The reason for both these rules is that with the lessened secretion of the stomach albuminous substances are digested in the stomach either not at all or only with difficulty, and in consequence mechanically irritate the bowel, causing discomfort.

Butter is not only permissible, but even indicated in all secretory anomalies.

Should there be periods of rest—*i. e.*, fluid diet—in the regimen? Although this may be resorted to for a short while, it hardly seems to be of much value, for in all these cases the primary seat of the disease is not in the stomach, and very little would be gained by special resting of the stomach. On the contrary, it seems to be more rational to regulate the diet in such a manner that it will gradually increase the work of the organs of digestion, so that the system becomes accustomed to digesting the usual foods.

The direct therapeutics of the stomach (medicaments, electricity) are generally well known, and need not be referred to here. They serve the purpose of alleviating the ailments of the stomach or to remove them.

As the result of this paper the following conclusions might be formulated:

1. The secretory functional disturbances of the stomach are not based on a primary change in the mucous membrane of the stomach. They rather produce, if they last for a longer time, lesions of the mucosa of greater or less extent.

2. The diagnosis of carcinoma of the stomach may, under specially favorable conditions, be made from a piece of gastric mucosa if a direct invasion of the gland substance by epithelial cells can be observed.

3. Therapeutically, attention must be directed principally toward the improvement of the general body state, and only secondarily by means of special measures against any secretory anomalies that may be present.

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